SUMMARY OF CRITICISMS ON EPA RISK ASSESSMENT

The EPA Risk Assessment includes a selective review of the literature on mainstream smoke, coupled with a series of unfounded and demonstrably false assumptions.

- The Risk Assessment's "hazard identification" does not review published data on the physical and chemical characteristics of ETS and relies upon the assumption that mainstream smoke and ETS are similar substances.

- The EPA document fails to recognize that the tobacco smoke to which the nonsmoker is exposed differs both physically and quantitatively from mainstream smoke.[1-8]

  The Risk Assessment does not address these differences and does not attempt any chemical or quantitative comparison between ETS and mainstream smoke.

  ETS is a highly diluted, aged and chemically altered mixture of sidestream smoke (the smoke emitted from the burning end of the cigarette) and exhaled mainstream smoke. The chemical composition of this mixture changes as it ages and interacts with other materials present in the room air.

- The active smoking data presented by the EPA are not relevant to the suspected route of exposure for ETS or, given the

2025500970

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

physical and quantitative differences between mainstream smoke and ETS, to a proper characterization of ETS itself.

Indeed, previous scientific reviews of ETS, including reviews by the U.S. Surgeon General[9] and the National Academy of Sciences,[10] have cautioned against such comparisons, stressing that there are significant differences between ETS and mainstream smoke that make assumptions of equivalence scientifically unjustified.

EPA does not adequately discuss the available data from animal studies of ETS exposure.

- **The only animal data referred to in the Risk Assessment are from a single inhalation study on hamsters in which it was reported that animals exposed to mainstream smoke exhibited an increase in laryngeal tumors; several studies on lung implantation of tobacco smoke condensates; and several results from studies of mouse skin-painting of cigarette smoke condensates.**

- **Studies relating to laryngeal cancer and to the reported results from implantation or skin-painting studies have little relevance to either the expected route of exposure for ETS,**

2025500971

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

i.e., inhalation to the lung, or to the disease endpoint under discussion (lung cancer).

- Animal inhalation studies employing fresh sidestream smoke and ETS were similarly not discussed in the Risk Assessment.

All three publicly available studies to date have reported no statistically significant increased incidence of tumors in animals exposed to ETS compared with controls.[11-14]

Moreover, the Risk Assessment fails to recognize that none of the lifetime, "whole smoke" exposure studies on animals, originally designed to assess possible biological effects from mainstream smoke exposures, have reported the induction of lung carcinomas via inhalation.

The Risk Assessment fails to reference a number of actual studies comparing levels of mutagens and other genotoxic markers in the body fluids of exposed and non-exposed nonsmokers.[15-24]

- **The results of those studies suggest no statistically significant increases in mutagenic activity in the body fluids of nonsmokers exposed to realistic levels of ETS compared with nonsmokers who are not exposed.**

2025500972

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

- Moreover, the "carcinogenic agents" supposedly identified in tobacco smoke (e.g., the "list" of suspected carcinogens referred to in the Risk Assessment) either are not suspected pulmonary carcinogens or have not been unequivocally demonstrated as tumorigenic to human tissue or to the lung tissue of experimental animals.[25-27]

In summary, the Risk Assessment's failure to include and consider relevant animal and short-term test data on ETS renders its "hazard identification" section incomplete. The active smoking data presented in the chapter are not relevant to the suspected route of exposure for ETS or, given the physical and chemical differences between MS and ETS, to a proper characterization of ETS itself. In addition, experimental inhalation data on MS, SS and ETS in animal models, not addressed in the chapter, provide no support for the claim that ETS is causally related to an increased risk of lung cancer.

In order to obtain the conclusion of the Risk Assessment, namely a statistically significant estimate of risk, the EPA combined data from epidemiologic studies using a statistical technique known as meta-analysis. The EPA's meta-analysis is scientifically unsound for a number of reasons.

2025500973

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

- The EPA's meta-analysis violated a fundamental principle of meta-analysis: it did not aggregate similar data. For example, all of the studies employed different questionnaires and different assessments of past exposure; five of the studies failed to match cases with controls; and the criteria for control selection were different in each study.[28]

- If the EPA had sought to address the lack of statistical power in some of the studies, it could have placed less emphasis on those studies and more upon the individual studies with sufficient statistical power. The individual studies with sufficient power do not report overall point estimates that achieve statistical significance.

- The statistical power question notwithstanding, the EPA has not addressed the issue of the aggregation of dissimilar data in its meta-analysis. Moreover, even the "common thread" identified by the EPA as justification for meta-analysis, spousal smoking, is defined differently across studies.

The EPA relied on a cumulative risk estimate derived from the meta-analysis of 11 U.S. epidemiologic studies of spousal smoking for its classification of ETS as a known human carcinogen.[29-39]

2025500974

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

- Not one of those 11 studies originally reported an overall risk estimate for lung cancer that was statistically significant, i.e., chance was not effectively ruled out as an explanation for the reported association between spousal smoking and lung cancer in nonsmokers.

- Indeed, even when the EPA lowered the statistical confidence limits reported for the U.S. studies from the standard 95% to 90%, all but one of the 11 studies individually failed to achieve statistical significance.[33]

- The EPA also reviewed 19 other epidemiologic studies from seven countries other than the United States.[40-58] Of these 19 studies, 13 reported no statistically significant overall point estimate for spousal smoking and lung cancer.

- Put another way, of the 30 studies reviewed by the EPA in all, 24 (a full 80%) did not support the Agency's conclusion. How then, can the EPA justify its conclusion that the epidemiology supports the classification of ETS as a Group A carcinogen?

- Even positive point estimates (greater than 1.00) that do not achieve statistical significance are compatible with the null hypothesis of "no association" between spousal smoking and

2025500975

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

lung cancer. Reported overall point estimates in six non-U.S. studies are statistically significant, but the EPA did not ascertain whether or not these purported associations are due to some feature that is coincidental to "marriage to a smoker" (e.g., common diet, occupational exposures, socio-economic status and other lifestyle characteristics).

The EPA intentionally lowered the confidence interval reported in the individual epidemiologic studies from 95% to 90% in an apparent attempt to rule out chance, and thus obtain a statistically significant cumulative risk estimate in its meta-analysis of U.S. studies.

- A confidence interval measures the probability that a statistical association was obtained by chance. The net effect of lowering the confidence interval from 95% to 90% is to artificially double the likelihood of ruling out chance as a possible explanation.

- The EPA's use of a lower confidence interval cannot be justified scientifically in light of the following obervations:

  The EPA and other federal agencies have generally used 95% confidence intervals in other risk assessments that have relied upon epidemiologic studies.

2025500976

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

Virtually all of the ETS epidemiologic studies upon which the EPA relied originally reported 95% confidence intervals.

The EPA's 1990 draft risk assessment on ETS used 95% confidence intervals.

Lowering the confidence interval was apparently the only way the EPA could reach its conclusion of a statistically significant increased risk for the combined U.S. studies.

The ETS Risk Assessment does not discuss the effect that lowering the confidence interval had on the data.

- The magnitude of the effect of the EPA's manipulation of the data is revealed when the standard 95% confidence interval is used. A meta-analysis of the U.S. studies employing the EPA's assumptions and using a 95% confidence interval reports no statistically significant summary risk estimate.[59]

The EPA failed to include in its meta-analysis relevant data from two major studies on ETS.[60,61] Months prior to the release of the Risk Assessment, these two studies appeared in the scientific literature, examining the purported association between spousal smoking status and lung cancer.

2025500977

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

- Funded in part by the National Cancer Institute, the 1992 Brownson, et al., study is one of the largest studies ever conducted on ETS exposure and lung cancer incidence.[60] It was not included in the Risk Assessment.

- The EPA had access to the two studies well in advance of the release of the final Risk Assessment.

- If the two studies were included in the EPA's meta-analysis of U.S. studies, a statistically nonsignificant cumulative risk estimate for spousal smoking would be calculated, even at the 90% confidence interval used by the EPA.[59]

- The inclusion of both the studies in the meta-analysis should negate the classification of ETS as a Group A carcinogen, in that a cumulative risk estimate for the U.S. studies would not be statistically significant, even at the 90% confidence interval.[59] The Agency should have estimated no statistically significant increased risk for lung cancer, at either a 95% or a 90% confidence interval.

- A meta-analysis including the two recent studies was forwarded to the EPA more than one month prior to the release of the final Risk Assessment.[59] The EPA chose not to include those

2025500978

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

data in its statistical calculations in the final Risk Assessment.[62]

The EPA reported a cumulative point estimate via meta-analysis for ETS of 1.19, when a risk estimate of 1.0 indicates no increase or decrease in reported risk. Epidemiologists generally agree that any estimated risk of less than 2.0 should be deemed "weak."[63]

- A point estimate below 2.0, given the very nature of epidemiology, may be due to some factor or factors associated with marriage to a smoker (e.g., diet, lifestyle, occupation, socioeconomic status, etc.).[64-74]

- Epidemiologic studies have reported risk estimates for lung cancer in nonsmokers in excess of 2.0 for these factors. The scientific literature suggests that such estimates may be large enough to account completely for the reported association between spousal smoking status and lung cancer.

- The EPA did not address fundamental questions regarding the detection limits of an epidemiologic study, or whether confounding factors have been effectively ruled out.

Only six of the 30 epidemiologic studies considered by the EPA reported overall statistically significant associations between

2025500979

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

spousal smoking and lung cancer; moreover, all six were conducted outside the United States.[43,45,47,49,50,57]

- These studies, as noted above for the U.S. studies, employed inconsistent methods of design and analysis.

- More importantly, the EPA failed to investigate whether the statistically significant associations reported in those studies could be the result of confounding by potential risk factors. The EPA failed to consider the large body of literature independent of the spousal smoking studies that addresses such factors.[64-87]

- EPA took the position that it was necessary to identify a single confounder, applicable across all the studies. Given the disparate study populations, methods, and analysis employed in the 30 studies, the identification of a single confounder to explain the reported association between spousal smoking and lung cancer is an unwarranted and, indeed, an unscientific expectation.[74]

Moreover, the scientific literature identifies several sources of possible bias in the spousal smoking studies.[88-98] These include publication bias, recall (exposure) bias, disease misclassification and smoking status misclassification.

2025500980

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

The ETS Risk Assessment itself states (3-53) that "misclassification errors must be addressed when using questionnaires to assess ETS exposure." The statement refers to exposure misclassification bias, which, along with other potential sources of bias, was not adequately addressed in the Risk Assessment.

- The Risk Assessment acknowledges only <u>one</u> possible bias, smoking status misclassification, and adjusts for it by employing an unpublished scientific model that had not been subjected to external peer review.

  The model contains numerous mathematical and conceptual errors, including a misclassification rate that is not representative of the U.S. population.[99-100]

  If a realistic misclassification rate had been used, EPA's own meta-analysis would have reported no statistically significant association between spousal smoking status and lung cancer.

- Adjustments for misclassification and "background exposure" are not meaningful, insofar as these adjustments were not performed by the original authors on data from their respective studies.

2025500981

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

The "background adjustment" is questionable since it relies on selected exposure data.

Moreover, the background adjustment presumes a causal relation for spousal smoking and lung cancer.

In relying on the 30 spousal smoking studies, the EPA selectively chose which data to use. Specifically, the EPA limited examination to the reported association between spousal smoking status in females and lung cancer.

- Although 11 of the 30 epidemiologic studies considered by the EPA also assessed workplace exposures to ETS, the Risk Assessment did not address those data.[31,33,35,37-39,47,48,51,54,58]

Nine of the 11 spousal smoking studies that examined workplace exposures reported no statistically significant association with lung cancer.[31,35,37,39,47,48,51,54,58]

If the data on workplace exposure reported in the 11 studies are pooled in a meta-analysis such as the one conducted in the Risk Assessment, the summary risk estimate is not statistically significant.[101]

2025500982

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

- Similarly, ten of the 30 epidemiologic studies contained data regarding reported exposures to ETS during childhood. The Risk Assessment also failed to consider those data.[32-34,37,39,40,42,48,55,56]

  Nine of the ten studies examining reported exposures to ETS during childhood reported no statistically significant association for lung cancer in adult nonsmokers.[32-34,39,40,42,48,55,56]

- The failure of the EPA to consider those data was scientifically unjustified. If the EPA had reviewed the data on workplace exposure and childhood residential exposure, the Agency should have found that the data contradicted its own conclusions.

Reports on dioxin, diesel emissions and EMF, prepared by working groups of EPA's Science Advisory Board, rejected the use of epidemiologic data like those in the ETS risk assessment precisely because the reported risks were low, and because the epidemiologic studies were not based on actual exposure data.

- The SAB's Ad Hoc Panel on Dioxin (November 1989) criticized a Review Draft on dioxin for just those oversights.[102]

2025500983

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

The SAB Report noted that most of the epidemiologic studies evaluated for dioxin "do not provide definitive data" and should be classified as "inconclusive, due in most cases to design limitations such as inadequate power and inadequate exposure assessment."

The Report concluded that "without good exposure data, the epidemiologic studies are meaningless."

- The SAB Workshop Review Draft on diesel emissions stated:[103]

> An excess risk of lung cancer was observed in three out of seven cohort studies and six out of seven case-control studies. Of these studies, two cohort and two case-control studies observed a dose-response relationship using duration of employment as a surrogate for dose. However, due to the lack of actual data on exposure to diesel exhaust in these studies and other methodologic limitations such as lack of latency analysis etc., the evidence of carcinogenicity in humans is considered to be limited for diesel exhaust exposure. (emphasis added)

The SAB Working Group report on EMF observed:[104]

> The association between cancer occurrence and exposure to either ELF or RF fields is not strong enough to constitute a proven causal relationship, largely because the relative risks in the published reports have seldom exceeded 3.0 in both childhood residential exposures and in occupational situations. (emphasis added)

And:

> The consistently repeated pattern of lymphoma, leukemia, nervous system cancer and lymphoma in childhood studies and the ruling out of several confounding exposure factors in the Savitz, et al. (1988) study argue in favor of a causal link between these tumor types in children and exposure to ELF magnetic or electric fields. However,

2025500984

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

the fact that the odds ratios are small and in many cases not statistically significant indicates that the association may not be strong and therefore argues against a causal relationship. (emphasis added)

A central contention of the Risk Assessment is that purported similarities between active smoking (mainstream smoke) and nonsmoker exposure to ETS imply "biologic plausibility" of the claim that ETS is a Group A carcinogen.

- Of course, the argument by analogy from active smoking to the "biologic plausibility" of the alleged role of ETS in disease causation does not provide a scientific evaluation of the hypothesis on ETS. Instead, it simply suggests the compatibility of two hypotheses, one for active smoking and one for ETS, in relation to human disease.

- The "biologic plausibility" question should be addressed by reference to animal inhalation studies on ETS and short-term tests on humans for ETS.

  The published results from these studies are negative for ETS.

The EPA's "dose-response analysis" is limited to performing a "re-analysis" of exposure-response trends reported in the individual spousal smoking studies.

2025500985

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

- Data are presented in some of the spousal studies in such a way that permits comparison of exposure data (i.e., the number of cigarettes or duration of recalled exposure) with estimates of risk. Such data are amenable to what statisticians refer to as a "trend test."

- The EPA's analysis did not rely on the study data as reported. Instead, the authors of the Risk Assessment recalculated trends for dose-response by adding what statisticians call a "pseudo-datum" representing zero exposure.[105]

- The addition of the "pseudo-datum" converts the statistically non-significant dose-response trends originally reported in the individual studies into statistically significant trends.

- The use of this inappropriate procedure essentially permits the establishment of a dose-response trend based upon any single category of exposure greater than zero.[105]

- Such trends do not support the existence of a dose-response among exposed individuals, nor do they rule out the possibility that the claimed associations relied upon are attributable to confounding or other factors that are correlated with spousal smoking.

2025500986

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

- The EPA suggests that its trend tests using unexposed subjects provide "evidential support of a causal association." However, if the dose-response analysis is performed correctly (excluding unexposed subjects), no spousal smoking study reports a significant dose-response trend.[105,106]

The ETS Risk Assessment is not based on accurate or verifiable information concerning exposure to ETS.

- The EPA relied upon questionnaire data on exposure recall contained in the spousal smoking studies.

- The accuracy of that data depends on an individual's ability to recall past events, such as how much a husband smoked in the past 20 to 30 years.[88,89,92]

The Risk Assessment fails to discuss or reference much of the relevant literature on the physical and chemical properties of ETS (as a mixture distinct from mainstream or sidestream smoke).

- In addition, only a small number of actual ETS exposure studies available in the published literature are discussed, and none of these are integrated into the body of the Risk Assessment.

2025500987

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

- Many of the studies of exposure to ETS constituents under realistic conditions in public places, workplaces and homes omitted from the Risk Assessment report minimal exposures to ETS: these reported exposures do not support the conclusions of the Risk Assessment.[1,4-8,107-138]

- The initial public review draft for the ETS Risk Assessment (1990) did not even contain an exposure assessment.

  Comments submitted to the public docket for the 1990 draft risk assessment observed that the EPA had failed to provide an exposure assessment which considered data from the numerous published studies on actual levels of ETS constituents in the air of public places and workplaces.

- In apparent response to that criticism, the revised 1992 draft of the ETS Risk Assessment contained a chapter entitled "Estimation of Environmental Tobacco Smoke Exposure," but the chapter's authors failed to consider at least 35 pertinent exposure studies on ETS constituent levels in public places.

  Furthermore, during its review of the chapter in July of 1992, the EPA's Science Advisory Board rejected the chapter and returned it to the author for rewriting.

2025500988

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

Nevertheless, without either an exposure assessment or recourse to any of the data pertaining to it, the Science Advisory Board endorsed the EPA's estimate of exposure and risk for the entire U.S. population.

A revised chapter on ETS exposure occurs in the final ETS Risk Assessment; however, the studies and data therein are not integrated into the Risk Assessment.

While some reports suggest that cotinine is a reliable marker for exposure to tobacco smoke,[139-140] critical assessments demonstrate that cotinine cannot serve as an accurate measure of ETS exposure.[141-150]

- **It has been reported that individuals metabolize nicotine in different ways at different times and that elimination rates for cotinine vary among individuals.**

- **In addition, recent research suggests that diet may contribute to levels of nicotine and cotinine found in the body, thereby interfering with reported exposure levels from nicotine in the ambient air.[151]**

- **Scientists have also noted that different methods of analysis may influence final recorded levels of cotinine.[152]**

2025500989

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

- Finally, it has been reported that because nicotine is largely present in the gas phase of ETS, measurement levels of its metabolite, cotinine, do not reflect exposures to other constituents that may be present, for example, in the particulate phase of ETS.

- For these reasons, cotinine should not be regarded as a reliable quantitative measure of ETS exposure.

2025500990

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

REFERENCES

1. Baker, R. and Proctor, C., "The Origins and Properties of Environmental Tobacco Smoke," Env Int (16): 231-245, 1990.

2. Löfroth, G., et al., "Characterization of Environmental Tobacco Smoke," Env Sci Technol (23): 610-614, 1989.

3. Reasor, M. and Will, J., "Assessing Exposure to Environmental Tobacco Smoke: Is It Valid to Extrapolate from Active Smoke?," Journal of Smoking Related Disorders 2(1): 111-127, 1991.

4. Proctor, C. and Dymond, H., "The Measurement of ETS Through Adsorption/Desorption Procedures." In: Indoor Air Quality. H. Kasuga (ed.). Springer-Verlag, Berlin, Heidelberg, 82-89, 1990.

5. Nystrom, C., et al., "Assessing the Impact of Environmental Tobacco Smoke on Indoor Air Quality: Current Status." In: Proceedings of the ASHRAE Conference, IAQ '86. April 20-23, 1986, Atlanta, Georgia, 213-244, 1986.

6. Rawbone, R., "The Aging of Sidestream Tobacco Smoke Components in Ambient Environments." In: Indoor Air Quality. H. Kasuga (ed.). Springer-Verlag, Berlin, Heidelberg, 55-61, 1990.

7. Piade, J., et al., "Assessment of ETS Impact on Office Air Quality." In: Indoor Air Quality. H. Kasuga (ed.). Springer-Verlag, Berlin, Heidelberg, 112-119, 1990.

8. Scherer, G., et al., "Importance of Exposure to Gaseous and Particulate Phase Components of Tobacco Smoke in Active and Passive Smokers," Occup Env Health (62): 459-466, 1990.

9. U.S. Department of Health and Human Services, Public Health Service, Office on Smoking and Health, The Health Consequences of Involuntary Smoking: A Report of the Surgeon General. DHHS Publication No. (CDC) 87-8398, Washington, D.C., U.S. Government Printing Office, 1986.

10. Committee on Passive Smoking, Board of Environmental Studies and Toxicology, National Research Council, Environmental Tobacco Smoke, Measuring Exposures and Assessing Health Effects. National Academy Press, Washington, D.C., 7-8, 1986.

11. Adlkofer, F., et al., "Exposure of Hamsters and Rats to Sidestream Smoke of Cigarettes: Preliminary Results of a 90-Day-Inhalation Study," Indoor and Ambient Air Quality, eds. R. Perry and P. Kirk, London, Selper Ltd. 251-258, 1988.

2025500991

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

12. Haley, N., et al. "Uptake of Sidestream Smoke by Syrian Golden Hamsters," Toxicol Letters 35 (1987): 83-88.

13. Haley, N., "Sidestream Smoke Uptake by Syrian Golden Hamsters in an Inhalation Bioassay," Indoor Air '87: Proceedings of the 4th International Conference on Indoor Air Quality and Climate, Institute for Water, Soil and Air Hygiene, Berlin: 68-73, 1987.

14. Coggins, C. et al., "Subchronic Inhalation Study in Rats Using Aged and Diluted Sidestream Smoke from a Reference Cigarette," Inhalation Tox 5: 77-96, 1993.

15. Martin, F., et al., "Urinary Excretion of Hydroxy-Phenanthrenes After Intake of Polycyclic Aromatic Hydrocarbons," Environ Int (15): 41-47, 1989.

16. Hoepfner, H., et al., "Hydroxy-Phenanthrenes in the Urine of Non-Smokers and Smokers," Toxicology Letters (35): 67-71, 1987.

17. Scherer, G., "Quantitative and Qualitative Differences in Tobacco Smoke Uptake Between Active and Passive Smoking," Indoor and Ambient Air Quality, eds. R. Perry and P. Kirk, Selper Ltd., London: 189-194, 1988.

18. Scherer, G., et al., "Urinary Mutagens, Hydroxy-Phenanthrene, and Thioether Excretion After Exposure to Environmental Tobacco Smoke," Indoor Air Quality, ed. H. Kasuga, Springer-Verlag, Berlin Heidelberg: 138-146, 1990.

19. Holz, O., et al., "32P-postlabelling Analysis of DNA Adducts in Monocytes of Smokers and Passive Smokers," Int Arch Occup Environ Health, 62: 299-303, 1990.

20. Sorsa, M., et al., "Cytogenetic Effects of Tobacco Smoke Exposure Among Involuntary Smokers," Mutation Res 222(2): 111-116, 1989.

21. Husgafvel-Pursiainen, K., "Sister-Chromatid Exchange and Cell Proliferation in Cultured Lymphocytes of Passively and Actively Smoking Restaurant Personnel," Mutation Res 190: 211-215, 1987.

22. Scherer, G., et al., "Urinary Mutagenicity After Controlled Exposure to Environmental Tobacco Smoke (ETS)," Toxicology Letters 35(1): 135-140, 1987.

2025500992

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

23. Bombick, D., et al., "Assessment of the Biological Activity of Mainstream or Environmental Tobacco Smoke (ETS) Using a Cellular Smoke Exposure Technique," Abstracts of the Twenty-Second Annual Scientific Meeting of the Environmental Mutagen Society, Kissimmee, Florida: April 1991. Abstract.

24. Scherer, G., "Biomonitoring of Exposure to Potentially Genotoxic Substances from Environmental Tobacco Smoke," Environ Int (15): 49-56, 1989.

25. Aviado, D., "Suspected Pulmonary Carcinogens in Environmental Tobacco Smoke," Environ. Tech. Letters, 9: 539-544, 1988.

26. Aviado, D., "Health Effects of 50 Selected Constituents of Environmental Tobacco Smoke," Indoor Air Quality, ed. H. Kasuga, Springer-Verlag, Berlin Heidelberg: 383-389, 1990.

27. Rodgman, A., "Environmental Tobacco Smoke," Reg Tox and Pharm 16: 223-244, 1992.

28. Sterling, T., Commentary Submitted to U.S. EPA Re: "Health Effects of Passive Smoking: Assessment of Lung Cancer in Adults and Respiratory Disorders in Children," (EPA/600/6-90/006A - External Review Draft) September, 1990.

29. Brownson, R.C., Reif, J.S., Keefe, T.J., Ferguson, S.W., and Pritzl, J.A., "Risk Factors for Adenocarcinoma of the Lung," American Journal of Epidemiology 125(1): 25-34, 1987.

30. Buffler, P.A., Pickle, L.W., Mason, T.J., and Contant, C., "The Causes of Lung Cancer in Texas." In: Lung Cancer: Causes and Prevention. M. Mizell and P. Correa (eds.). Deerfield Beach, Verlag Chimie International, 83-99, 1984.

31. Butler, T.L., The Relationship of Passive Smoking to Various Health Outcomes Among Seventh-Day Adventists in California, Ph.D. Thesis, University of California, 1988.

32. Correa, P., Pickle, L.W., Fontham, E., Lin, Y., and Haenszel, W., "Passive Smoking and Lung Cancer," Lancet II: 595-597, 1983.

33. Fontham, E.T.H., Correa, P., "Wu-Williams, A., Reynolds, P., Greenberg, R.S., Buffler, P.A., Chen, V.W., Boyd, P., Alterman, T., Austin, D.F., Liff, J., and Greenberg, S.D., "Lung Cancer in Nonsmoking Women: A Multicenter Case-Control Study," Cancer Epidemiology, Biomarkers & Prevention 1: 35-43, 1991.

2025500993

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

34. Garfinkel, L., "Time Trends in Lung Cancer Mortality Among Nonsmokers and a Note on Passive Smoking," Journal of the National Cancer Institute 66(6): 1061-1066, 1981.

35. Garfinkel, L., Auerbach, O., and Joubert, L., "Involuntary Smoking and Lung Cancer: A Case-Control Study," Journal of the National Cancer Institute 75(3): 463-469, 1985.

36. Humble, C.G., Samet, J.M., and Pathak, D.R., "Marriage to a Smoker and Lung Cancer Risk," American Journal of Public Health 77(5): 598-602, 1987.

37. Janerich, D., Thompson, W.D., Varela, L.R., Greenwald, P., Chorost, S., Tucci, C., Zaman, M.B., Melamed, M.R., Kiely, M., and McKneally, M.F., "Lung Cancer and Exposure to Tobacco Smoke in the Household," New England Journal of Medicine 323: 632-636, 1990.

38. Kabat, G., and Wynder, E., "Lung Cancer in Nonsmokers," Cancer 53(5): 1214-1222, 1984.

39. Wu, A., Henderson, B.E., Pike, M.C., and Yu, M.C. "Smoking and Other Risk Factors for Lung Cancer in Women," Journal of the National Cancer Institute 74(4): 747-751, 1985.

40. Akiba, S., Kato, H., and Blot, W.J., "Passive Smoking and Lung Cancer Among Japanese Women," Cancer Research 46: 4804-4807, 1986.

41. Chan, W.C., and Fung, S.C., "Lung Cancer in Non-Smokers in Hong Kong." In: Cancer Campaign Vol. 6 Cancer Epidemiology. E. Grundmann (ed.). Stuttgart, Gustav Fischer Verlag, 199-202, 1982.

42. Gao, Y.-T., Blot, W.J., Zheng, W., Ershow, A.G., Hsu, C.W., Levin, L.I., Zhan, R., and Fraumeni, J.F., "Lung Cancer Among Chinese Women," International Journal of Cancer 40: 604-609, 1987.

43. Geng, G.-Y., Liang, T.H., Zhang, A.Y., and Wu, G.L., "On the Relationship Between Smoking and Female Lung Cancer." In: Smoking and Health 1987. M. Aoki, S. Hisamichi and S. Tominaga (eds.). Amsterdam, Excerpta Medica, 483-486, 1988.

44. Gillis, C.R., Hole, D.J., Hawthorne, V.M., and Boyle, P., "The Effect of Environmental Tobacco Smoke in Two Urban Communities in the West of Scotland." In: ETS - Environmental Tobacco Smoke: Report from a Workshop on Effects and Exposure Levels. R. Rylander, Y. Peterson and M.-C. Snella (Eds.).

2025500994

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

Published simultaneously in European Journal of Respiratory Disease, Supplement 133, Volume 65, 121-126, 1984.

45. Hirayama, T., "Non-Smoking Wives of Heavy Smokers Have a Higher Risk of Lung Cancer: A Study from Japan," British Medical Journal 282: 183-185, 1981.

46. Inoue, R., and Hirayama, T., "Passive Smoking and Lung Cancer in Women." In: Smoking and Health 1987. M. Aoki, S. Hisamichi and S. Tominaga (eds.). Amsterdam, Exerpta Medica, 283-284, 1988.

47. Kalandidi, A., Katsouyanni, K., Voropoulou, N., Bastas, G., Saracci, R., and Trichopoulos, D., "Passive Smoking and Diet in the Etiology of Lung Cancer Among Non-Smokers," Cancer Causes and Control 1: 15-21, 1990.

48. Koo, L.C., Ho, J.H.-C., Saw, D. and Ho. C.Y., "Measurements of Passive Smoking and Estimates of Lung Cancer Risk Among Non-Smoking Chinese Females," International Journal of Cancer 39: 162-169, 1987.

49. Lam, W.K., A Clinical and Epidemiological Study of Carcinoma of Lung in Hong Kong, M.D. Thesis, University of Hong Kong, 1985.

50. Lam, T.H., Kung, I.T.M., Wong, C.M., Lam, W.K., Kleevens, J.W.L., Saw, D., Hsu, C., Seneviratne, S., Lam, S.Y., Lo, K.K., and Chan, W.C., "Smoking, Passive Smoking and Histological Types in Lung Cancer in Hong Kong Chinese Women," British Journal of Cancer 56(5): 673-678, 1987.

51. Lee, P.N., Chamberlain, J., and Alderson, M.R., "Relationship of Passive Smoking to Risk of Lung Cancer and Other Smoking-Associated Diseases," British Journal of Cancer 54: 97-105, 1986.

52. Liu, Z., He, X., and Chapman, R.S., "Smoking and Other Risk Factors for Lung Cancer in Xuanwei, China," International Journal of Epidemiology 20(1): 26-31, 1991.

53. Pershagen, G., Hrubec, Z., and Svensson, C., "Passive Smoking and Lung Cancer in Swedish Women," American Journal of Epidemiology 125(1): 17-24, 1987.

54. Shimizu, H., Morishita, M., Mizuno, K., Masuda, T., Ogura, Y., Santo, M., Nishimura, M., Kunishima, K., Karasawa, K., Nishiwaki, K., Yamamoto, M., Hisamichi, S., and Tominaga, S., "A Case-Control Study of Lung Cancer in Nonsmoking Women," Tohoku Journal of Experimental Medicine 154: 389-397, 1988.

2025500995

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

55. Sobue, T., Suzuki, R., Nakayama, N., Inubuse, C., Matsuda, M., Doi, O., Mori, T., Furuse, K., Fukuoka, M., Yasumitsu, T., Kuwabara, O., Ichigaya, M., Kurata, M., Kuwabara, M., Nakahara, K., Endo, S., and Hattori, S., "Passive Smoking Among Nonsmoking Women and the Relationship Between Indoor Air Pollution and Lung Cancer Incidence -- Results of a Multicenter Case Controlled Study," Gan to Rinsho 36(3): 329-333, 1990.

56. Svensson, C., Pershagen, G., and Klominek, J., "Smoking and Passive Smoking in Relation to Lung Cancer in Women," Acta Oncologica 28(5): 623-639, 1989.

57. Trichopoulos, D., Kalandidi, A., Sparros, L., and MacMahon, B., "Lung Cancer and Passive Smoking," International Journal of Cancer 27(1): 1-4, 1981.

58. Wu-Williams, A.H., Dai, X.D., Blot, W., Xu, Z.Y., Sun, X.W., Xiao, H.P., Stone, B.J., Yu, S.F., Feng, Y.P., Ershow, A.G., Sun, J., Fraumeni, J.F., and Henderson, B.E., "Lung Cancer Among Women in North-East China," British Journal of Cancer 62: 982-987, 1990.

59. Tozzi, J.J., Letter to Dr. Erich Bretthauer, Assistant Administrator for Research and Development, U.S. EPA, December 4, 1992.

60. Brownson, R.C., Alavanja, M.C.R., Hock, E.T., and Loy, T.S., "Passive Smoking and Lung Cancer in Nonsmoking Women," American Journal of Public Health 82(11): 1525-1530, 1992.

61. Stockwell, H.G., Goldman, A.L., Lyman, G.H., Noss, C.I., Armstrong, A.W., Pinkham, P.A., Candelora, E.C., and Brusa, M.R., "Environmental Tobacco Smoke and Lung Cancer Risk in Nonsmoking Women," Journal of the National Cancer Institute 84(18): 1417-1422, 1992.

62. Bretthauer, E., U.S. EPA, Correspondence to Jim J. Tozzi, December 17, 1992.

63. Wynder, E.L. and Kabat, G.C., "Environmental Tobacco Smoke and Lung Cancer: A Critical Assessment." In: Indoor Air Quality. H. Kasuga (ed.). Berlin, Heidelberg, Springer-Verlag, 5-15, 1990.

64. Koo, L.C., Ho, J.H.-C. and Rylander, R., "Life-History Correlates of Environmental Tobacco Smoke: A Study on Nonsmoking Hong Kong Chinese Wives with Smoking Versus Nonsmoking Husbands," Soc Sci Med 26(7): 751-760, 1988.

2025500996

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

65. Koo, L., "Dietary Habits and Lung Cancer Risk Among Chinese Females in Hong Kong Who Never Smoked," Nutrition and Cancer 11: 155-172, 1988.

66. Koo, L.C., "Environmental Tobacco Smoke and Lung Cancer: Is It the Smoke or the Diet?" In: Present and Future of Indoor Air Quality. C.J. Bieva, Y. Courtois and M. Govaerts (eds.). Amsterdam, Elsevier Science Publishers, 65-75, 1989.

67. Koo, L.C. and Ho, J.H.-C., "Is There a Threshold Effect for ETS? Results of Data from Chinese Females Who Had Never Smoked." In: Indoor Air Quality. H. Kasuga (ed.). Berlin, Heidelberg, Springer-Verlag, 290-298, 1990.

68. Sidney, S., Caan, B.J., and Friedman, G.D., "Daily Intake of Carotene in Nonsmokers With and Without Passive Smoking at Home," American Journal of Epidemiology 129(6): 1305-1309, 1989.

69. Le Marchand, L., Wilkens, L.R., Hankin, J.H. and Haley, N.J., "Dietary Patterns of Female Nonsmokers With and Without Exposure to Environmental Tobacco Smoke," Cancer Causes and Control 2: 11-16, 1991.

70. Waller, K.D., and Smith, A.H., "Effects of Socioeconomic Status and Smoking on Serum Beta-Carotene," Archives of Environmental Health 46(2): 120, 1991.

71. Rylander, R., "Environmental Tobacco Smoke and Lung Cancer," The New England Journal of Medicine 323(12): 834, 1990.

72. Sterling, T., Weinkam, J. and Sterling, D., "Exposure of Homemakers to Toxic Contaminants: I. Differences in Chronic Conditions Between Homemakers and Employed Persons." In: Indoor Air '90: Proceedings of the 5th International Conference on Indoor Air Quality and Climate. Toronto, Canada, Volume 1, 471-476, July 29-August 3, 1990.

73. Gori, G.B., and Mantel, N., "Mainstream and Environmental Tobacco Smoke," Regulatory Toxicology and Pharmacology 14: 88-105, 1991.

74. Butler, W., "Review of: Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders," (EPA/600/6-90/006B; SAB Review Draft; May 1992). Submission to EPA-SAB-IAQTHE Committee, July 21, 1992.

75. Mumford, J.L., He, X.Z., Chapman, R.S., Cao, S.R., Harris, D.B., Li, X.M., Xian, Y.L., Jiang, W.Z., Xu, C.W., Chuang,

2025500997

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

J.C., Wilson, W.E. and Cooke, M., "Lung Cancer and Indoor Air Pollution in Xuan Wei, China," Science 235: 217-220, 1987.

76. Chapman, R.S., Mumford, J.L., Harris, D.B., He, X., Jiang, W. and Yang, R., "the Epidemiology of Lung Cancer in Xuan Wei, China: Current Progress, Issues and Research Strategies," Archives of Environmental Health 43(2): 180-185, 1988.

77. Wang, F.-L., et al., "Analysis of Risk Factors for Female Lung Cancer in Haerbin: Indoor Air Pollution," Chinese Journal of Preventive Medicine 23(5): 270-273, 1989.

78. Du, Y.-X. and Ou, X.-L., "Indoor Air Pollution and Woman Lung Cancer." In: Indoor Air '90: Proceedings on the 5th International Conference on Indoor Air Quality and Climate. Toronto, Canada, Volume 1, 59-64, July 29-August 3, 1990.

79. He, F., "Health Effects of Indoor Air Pollution by Coal Combustion in China." In Indoor Air Quality and Ventilation. London, Selper, Ltd., 305-311, 1990.

80. Xu, Z.-Y., Blot, W.J., Xiao, H.-P., Wu, A., Feng, Y.-P., Stone, B.J., Sun, J., Ershow, A.G., Henderson, B.E. and Fraumeni, J.F., "Smoking, Air Pollution, and the High Rates of Lung Cancer in Shenyang, China," Journal of the National Cancer Institute 81: 1800-1806, 1989.

81. Bu, W., "Ventilation and Its Effects on Pollution in Chinese Kitchens." In: Indoor Air Quality and Ventilation. London, Selper, Ltd., 129-132, 1990.

82. Basas, B., "An Overview of Indoor Air Quality in Certain Greek Industries, Workshops and Offices." In: Indoor Air Quality and Ventilation. London, Selper, Ltd., 237-240, 1990.

83. Katzenstein, A.W., "An Estimate of Adult Mortality in the United States from Passive Smoking: A Response," Environment International 16: 175-178, 1990.

84. Chen, B.H., Hong, C.J., Pandey, M.R. and Smith, K.R., "Indoor Air Pollution in Developing Countries," World Health Statistics Quarterly 43: 127-138, 1990.

85. Tewes, F.J., Koo, K.C., Meisgen, T.J. and Rylander, R., "Lung Cancer Risk and Mutagenicity of Tea," Environmental Research 52: 23-33, 1990.

86. Rylander, R. and Koo, L.C., "Environmental Tobacco Smoke and Lung Cancer -- Recent Aspects on Confounders and Dose Levels."

2025500998

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

In: Indoor Air Quality. H. Kasuga (ed.). Berlin, Heidelberg, Springer-Verlag, 133-137, 1990.

87. Holst, P.A., Kromhout, D. and Brand, R., "For Debate: Pet Birds as an Independent Risk Factor for Lung Cancer," British Medical Journal 297: 1319-1321, 1988.

88. Coultas, D., et al., "Questionnaire Assessment of Lifetime and Recent Exposure to Environmental Tobacco Smoke," American Journal of Epidemiology 130(2): 338-347, 1989.

89. Coultas, D., et al., "Variability of Measures of Exposure to Environmental Tobacco Smoke in the Home," American Review of Respiratory Disease 142: 602-606, 1990.

90. Coultas, D., et al., "A Personal Monitoring Study to Assess Workplace Exposure to Environmental Tobacco Smoke," American Journal of Public Health 80(8): 988-990, 1990.

91. Friedman, G., et al., "Prevalence and Correlates of Passive Smoking," American Journal of Public Health 73(4): 401-405, 1983.

92. Pron, G., et al., "The Reliability of Passive Smoking Histories Reported in a Case-Control Study of Lung Cancer," American Journal of Epidemiology 127(2): 267-273, 1988.

93. National Academy of Sciences, National Research Council, Committee on Advances in Assessing Human Exposure to Airborne Pollutants, Human Exposure Assessment for Airborne Pollutants: Advances and Opportunities, National Academy Press, Washington, D.C., 215-216, 1991.

94. Schenker, M., Hammond, K., Samuels, S., Kado, N. and Woskie, S., "Assessment of Environmental Tobacco Smoke Exposure in Epidemiologic Studies," Chest 91(2): 313-314, 1987. Abstract.

95. Lerchen, M.L. and Samet, J.M., "An Assessment of the Validity of Questionnaire Responses Provided by a Surviving Spouse," American Journal of Epidemiology 123(3): 481-489, 1986.

96. Wynder, E.L. and Kabat, G.C., "Environmental Tobacco Smoke and Lung Cancer: A Critical Assessment." In: Indoor Air Quality. H. Kasuga (ed.). Berlin, Heidelberg, Springer-Verlag, 5-15, 1990.

97. Kilpatrick, S., "Misclassification of Environmental Tobacco Smoke Exposure: Its Potential Influence on Studies of Environmental Tobacco Smoke and Lung Cancer," Toxicology Letters 35: 163-168, 1987.

2025500999

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

98. Faccini, J.M., "The Role of Histopathology in the Evaluation of Risk of Lung Cancer from Environmental Tobacco Smoke," Experimental Pathology 37: 177-180, 1989.

99. Sears, S., et al., "Comments on Chapter 5 and Appendix B of the EPA Document: 'Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders,'" EPA/600/6-90/006B. Submission to the EPA-SAB-IAQTHE Committee, July 21, 1992.

100. Lee, P., "'Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders': A Commentary on Issues Relating to Lung Cancer in the May 1992 SAB Review Draft," Submission to the EPA-SAB-IAQTHE Committee, July 1, 1992.

101. Layard, M. "Comments on the EPA Review Draft: Respiratory Effects of Passive Smoking: Lung Cancer and Other Disorders," Submitted to EPA-SAB-IAQTHE Committee, June, 1992.

102. SAB Ad Hoc Panel on Dioxin, "Panel Report: Review Draft on Dioxin," U.S. EPA, November, 1989.

103. SAB Panel on Diesel Emissions, "Health Assessment Document for Diesel Emissions, "Workshop Review Draft EPA/600/8-90/57A, July, 1990.

104. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Evaluation of the Potential Carcinogenicity of Electromagnetic Fields, EPA/600/6-90/005A, Workshop Review Draft, June, 1990.

105. Switzer, P., "Comments on EPA May 1992 Draft Report: 'Respiratory Health Effects of Passive Smoking. . .' Presentation to the EPA/SAB Review Panel Meeting, July 21, 1992.

106. Tweedie, R., "Lung Cancer and Exposure to Environmental Tobacco Smoke. Assessment of Issues Raised in the Review Draft of the Environmental Protection Agency of the United States," September 18, 1990.

107. Sterling, T., et al., "Indoor Byproduct Levels of Tobacco Smoke: A Critical Review of the Literature," J Air Pollut Control Assoc 32(3): 250-259, 1982.

108. Sterling, T., et al., "Environmental Tobacco Smoke and Indoor Air Quality in Modern Office Work Environments," J Occup Med 29(1): 57-62.

2025501000

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

109. Pedelty, J. and Holcomb, L., "A Computer Simulation of Indoor Air Quality Which Models Changes in Point Sources and Ventilation," Environ Technol Letters 11: 1053-1062, 1990.

110. Adlkofer, F. "Biological Effects After Exposure to ETS." In: Indoor Air Quality. Buenos Aires, The National Academy of Sciences of Buenos Aires, 61-78, 1989.

111. Kirk, P., et al., "Environmental Tobacco Smoke in Indoor Air." In: Indoor and Ambient Air Quality. R. Perry and P. Kirk (eds.). London, Selper Ltd., 99-112, 1988.

112. Carson, J. and Erikson, C., "Results from Survey of Environmental Tobacco Smoke in Offices in Ottawa, Ontario," Environ Technol Letters 9: 501-508, 1988.

113. Sterling, T. and Mueller, B., "Concentrations of Nicotine, RSP, CO and CO_2 in Nonsmoking Areas of Offices Ventilated by Air Recirculated from Smoking Designated Areas," Am Ind Hyg Assoc J 49(9): 423-426, 1988.

114. Cox, B. and Whichelow, M., "Carbon Monoxide Levels in the Breath of Smokers and Nonsmokers: Effect of Domestic Heating Systems," J Epidemiol Community Health 39: 75-78, 1985.

115. Good, B., et al., "Effect of Cigarette Smoking on Residential NO_2 Levels," Environ Int 8: 167-175, 1982.

116. Godish, T., "Formaldehyde Exposures from Tobacco Smoke: A Review," AJPH 79(8): 1044-1045, 1989.

117. Proctor, C., "The Analysis of the Contribution of ETS to Indoor Air." In: Indoor and Ambient Air Quality. R. Perry and P. Kirk (eds.). London, Selper Ltd., 57-66, 1988.

118. Eatough, D., et al., "Assessing Exposure to Environmental Tobacco Smoke." In Indoor and Ambient Air Quality. R. Perry and P. Kirk (eds.). London, Selper Ltd., 131-140, 1988.

119. Proctor, C., et al., "Measurements of Environmental Tobacco Smoke in an Air-Conditioned Office Building," Environ Technol Letters (10): 1003-1018, 1989.

120. Proctor, C., "A Multi-Analyte Approach to the Measurement of Environmental Tobacco Smoke." In: Indoor Air quality and Ventilation. F. Lunau and G. Reynolds (eds.). London, Selper Ltd. 427-436, 1990.

121. Jenkins, R., et al., "Development and Application of a Thermal Desorption-Based Method for the Determination of Nicotine in

2025501001

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

Indoor Environments." In: Indoor and Ambient Air Quality. R. Perry and P. Kirk (eds.). London, Selper Ltd., 493-496, 1988.

122. Muramatsu, M., et al., "Estimation of Personal Exposure to Ambient Nicotine in Daily Environment," Arch Occup Environ Health 59: 545-550, 1987.

123. Thompson, C., et al., "A Thermal Desorption method for the Determination of Nicotine in Indoor Environments," Environ Sci Technol 23: 529-435, 1989.

124. Hosein, R., "The Relationship Between Pollutant Levels in Homes and Potential Sources." In: Transactions: Indoor Air Quality in Cold Climates, Hazards and Abatement Measures. D. Walkinshaw (ed.). Pittsburgh, Air Pollution Control Association, 250-260, 1986.

125. Quackenboss, J. and Lebowitz, M., "Indoor-Outdoor Relationships for Particulate Matter: Exposure Classifications and Health Effects," Environ Int 15: 353-360, 1989.

126. Sterling, T., "ETS Concentrations Under Different Conditions of Ventilation and Smoking Regulation." In: Indoor and Ambient Air Quality. R. Perry and P. Kirk (eds.). London, Selper Ltd., 89-98, 1988.

127. First, M., "Constituents of Sidestream and Mainstream Tobacco Smoke and Markers to Quality Exposure to Them." In: Indoor Air and Human Health. Chelsea, Michigan, Lewis Publishers, 195-203, 1985.

128. Bouhuys, A., et al., "Do Present Levels of Air Pollution Outdoors Affect Respiratory Health?," Nature 276: 466-471, 1978.

129. Binder, R., et al., "Importance of the Indoor Environment in Air Pollution Exposure," Arch Environ Health 31(6): 277-279, 1976.

130. Lebowitz, M., et al., "Respiratory Symptoms and Peak Flow Associated with Indoor and Outdoor Air Pollutants in the Southwest," J. Air Pollut Control Assoc 35: 1154-1158, 1985.

131. McAughey, J., et al., "Risk Assessment of Exposure to Indoor Air Pollutants," Env Int (11): 295-302, 1990.

132. Stehlik, G., et al., "Concentration of Dimethylnitrosamine in the Air of Smoke-Filled Rooms," Ecotoxicol Environ Safety 6: 495-500, 1982.

2025501002

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

133. Sega, K., and Fugas, M., "Nitrogen Dioxide Concentrations in Residences." In: Indoor and Ambient Air Quality. R. Perry and P. Kirk (eds.). London, Selper Ltd., 493-496, 1988.

134. Bayer, C. and Black, M., "Thermal Desorption/Gas Chromatographic/Mas Spectrometric Analysis of Volatile Organic Compounds in the Offices of Smokers and Nonsmokers," Biomed and Envir Mass Spect 14(8): 363-367, 1987.

135. Adlkofer, F., et al., "Significance of Exposure to Benzene and Other Toxic Compounds Through Environmental Tobacco Smoke," J Cancer Res Clin Oncol (116): 591-598, 1990.

136. Godish, T., "Residential Formaldehyde: Increased Exposure Levels Aggravate Adverse Health Effects," J. of Environ Health 53(3): 34-35, 1990.

137. Hugod, C., et al., "Exposure of Passive Smokers to Tobacco Smoke Constituents," Int Arch Occup Environ Health (42): 21-29, 1978.

138. Proctor, C., et al., "Measurement of Environmental Tobacco Smoke in an Air-Conditioned Office Building." In: Present and Future of Indoor Air Quality. C.J. Bieva, et al. (eds.). Brussels, Elsevier Science Publishers, 169-172, 1989.

139. Wald, N., et al., "Urinary Cotinine as A Marker of Breathing Other People's Tobacco Smoke," Lancet I: 230-231, 1984.

140. Watts, R., et al., "Cotinine Analytical Workshop Report: Consideration of Analytical Methods for Determining Cotinine in Human Body Fluids as a Measure of Passive Exposure to Tobacco Smoke," Environ Health Pers 84: 173-182, 1990.

141. Curvall, M., et al., "Simulation and Evaluation of Nicotine Intake During Passive Smoking: Cotinine Measurements in Body Fluids of Nonsmokers Given Intravenous Infusions of Nicotine," Clin Pharmacol Ther 47(1): 42-49, 1990.

142. Itani, S., et al., "A Comparison of Plasma and Urinary Nicotine and Cotinine Levels in Smokers and Nonsmokers: Nicotine Excretion Pathways Are Possibly Differential According to the Dosage of Tobacco Smoke Uptake." In: Indoor Air Quality. H. Kasuga (ed.). Springer-Verlag, Berlin, Heidelberg, 202-212, 1990.

143. Idle, J., "Commentary: Titrating Exposure to Tobacco Smoke Using Cotinine -- A Minefield of Misunderstanding," Journal of Clinical Epidemiology 43(4): 313-317, 1990.

2025501003

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000

144. Biber, A., et al., "Determination of Nicotine and Cotinine in Human Serum and Urine: An Interlaboratory Study," Toxicology Letters 35(1): 45-52, 1987.

145. Haley, N., et al., "Elimination of Cotinine from Body Fluids: Disposition in Smokers and Nonsmokers," AJPH 79(8): 1046-1048, 1989.

146. Cummings, K., et al., "Measurement of Current Exposure to Environmental Tobacco Smoke," Archives of Environ Health 45(2): 74-79, 1990.

147. Lee, P., "Lung Cancer and Passive Smoking," Br J Cancer 63: 161-162, 1991.

148. Johnson, L., et al., "Passive Smoking Under Controlled Conditions," Int Arch Occup Environ Health 56: 99-110, 1985.

149. Letzel, H., et al., "Measuring Problems in Estimating the Exposure to Passive Smoking Using the Excretion of Cotinine," Toxicology Letters 35(1): 35-44, 1987.

150. Schwartz, S., et al., "Mathematical Modelling of Nicotine and Cotinine as Biological Markers of Environmental Tobacco Smoke Exposure," Toxicology Letters 35(1): 53-58, 1987.

151. Davis, R., et al., "Dietary Nicotine: A Source of Urinary Cotinine," Fd Chem Toxic 29(12): 821-827, 1991.

152. Adlkofer, F., et al., "Passive Smoking," New England Journal of Medicine 312(11): 719-720, 1984.

2025501004

Source: https://www.industrydocuments.ucsf.edu/docs/zzjj0000